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Planning management and delivery of the growth-restricted fetus

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A B S T R A C T

A uniform approach to management of fetal growth restriction (FGR) improves outcome, prevents stillbirth, and allows appropriately timed delivery. An estimated fetal weight below the tenth percentile with coexisting abnormal umbilical artery (UA), middle cerebral artery (MCA), or cerebroplacental ratio Doppler index best identifies the small fetus requiring surveillance. Placental perfusion defects are more common earlier in gestation; accordingly, early-onset (≤32 weeks of gestation) and late-onset (>32 weeks) FGR differ in clinical phenotype. In early-onset FGR, progression of UA Doppler abnormality determines clinical acceleration, while abnormal ductus venosus (DV) Doppler precedes deterioration of biophysical variables and stillbirth. Accordingly, late DV Doppler changes, abnormal biophysical variables, or an abnormal cCTG require delivery. In late-onset FGR, MCA Doppler abnormalities precede deterioration and stillbirth. However, from 34 to 38 weeks, randomized evidence on optimal delivery timing is lacking. From 38 weeks onward, the balance of neonatal versus fetal risks favors delivery.

Introduction

Fetal growth restriction (FGR) is a physical manifestation of a number of etiologies including placental dysfunction. The key issues in the management of a pregnancy complicated by FGR are the

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identification of the fetus at greatest risk for deterioration, the utilization of the most appropriate surveillance approach, and determination of the delivery threshold. There is good evidence that a uniform management to diagnosis and management of FGR consistently produces better outcome than is reported in observational studies that rely on a range of diagnostic, surveillance, and delivery criteria [1–6]. One important challenge that affects all aspects of management is the phenotypic variation of FGR across gestational age. The aim of this article is to review the clinical phenotype of placenta-based FGR and the main aspects of the management components in these patients.

Clinical phenotype of fetal growth restriction and gestational age

FGR evolves from a preclinical phase to clinically apparent growth delay and may eventually progress to fetal deterioration. Normal fetal growth and development relies on the placental delivery of nutrients, as well as transplacental gas and fluid exchange [7]. Decreased transplacental glucose and nutrient transfer, due to a reduction in either active transport mechanisms or abnormal placental perfusion, can lead to FGR. Because hepatic glycogen stores are depleted under these circumstances, growth of the abdominal circumference (AC) decelerates. A decrease in transplacental fluid transfer or impaired fetal fluid uptake in the setting of abnormal umbilical venous blood flow may accompany or predate nutritional deficiency and is associated with oligohydramnios [8–10]. Increased blood flow resistance in the maternal uterine arteries [11–13] or the fetal umbilical arteries [14] indicates that the vascular mechanisms that are important for maternal nutrient delivery or fetal nutrient uptake and waste exchange are deficient. When transplacental gas transfer becomes abnormal, leading first to hypoxemia and then hypercarbia, additional fetal responses such as decreased activity [15,16], decreased blood flow resistance [17,18], or increased peak systolic velocity [19,20] in the middle cerebral artery (MCA) may be observed. The aspects of placental function, which are predominantly affected, determine the clinical phenotype of FGR at the time of diagnosis and progression as placental dysfunction worsens. Placental lesions that are associated with underperfusion in the fetal and maternal compartments are more common at earlier gestational age [21–23]. Although placental pathology probably changes over the continuum of gestational age, expert opinion considers FGR presenting before 32 weeks as “early onset” and thereafter as “late onset” [24].

Early-onset FGR

With a higher prevalence of villous perfusion abnormalities, decreased umbilical artery (UA) end-diastolic velocity (EDV) proportional to the degree of flow impairment is more commonly observed in early-onset FGR [25,26]. It is the rate of increase in UA blood flow resistance and specifically how rapidly EDV is lost that determines the rate and degree of fetal deterioration [26–28]. In the preterm FRG fetus with increased UA blood flow resistance, MCA brain sparing may be present or develop as a sign of progressive cardiovascular responses to placental dysfunction [26,29–31]. Further increasing UA blood flow resistance, worsening acidemia, and superimposing cardiac dysfunction eventually can lead to abnormal precordial venous Doppler parameters [26,27,30,32,33]. This degree of cardiovascular deterioration typically precedes an abnormal biophysical profile score or stillbirth (Fig. 1) [33–35]. The latency from diagnosis to late cardiovascular changes may range between 4 and 6 weeks and is determined by the rate at which UA-EDV is lost and by the gestational age [26,36].

Late-onset FGR

Owing to the higher prevalence of villous diffusion abnormalities and a lesser degree of perfusion abnormalities, late-onset FGR may present with little or no UA index elevation but rather uterine artery Doppler index increase or a decrease in umbilical venous volume flow [37]. Despite the seemingly “normal” placental function in the presence of a normal UA Doppler index, MCA brain sparing or a decrease in the cerebroplacental Doppler ratio (CPR) may be observed documenting decreased placental O_2 transfer [26,30,35,38,39]. In the clinical progression, a decreased CPR progressing to brain sparing can be observed [40]. Additional signs of deterioration preceding stillbirth include a decline in amniotic fluid volume or abnormal fetal heart rate parameters (Fig. 2) [30,35]. Because UA Doppler is